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The Effect of Heat Acclimation on Hand Cooling Efficacy During Exercise Heat Stress

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The Effect of Heat Acclimation on Hand Cooling Efficacy During Exercise Heat Stress

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ABSTRACT

The Effect of Heat Acclimation on Hand Cooling Efficacy During Exercise Heat Stress

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CONTEXT: Heat acclimation (HA) and body cooling during exercise in the heat both help to mitigate the rise in body temperature; however, the effects of HA and hand cooling combined remain unexplored. **OBJECTIVE:** To determine the separate and combined effects of HA and hand cooling efficacy, during and after exercise in the heat. **DESIGN:** Randomized, counterbalanced, crossover design. **SETTING:** Research laboratory. **PARTICIPANTS:** Seventeen non-HA (NHA) males (mean±SE; age, 23±1y; height, 179.5±1.6cm; mass, 75.30±2.27kg; VO_2 max, 54.1±1.3 $ml \cdot kg^{-1} \cdot min^{-1}$) completed 2 pre-HA heat stress tests (HST1 and HST2), 10 days of HA, and 2 post-HA heat stress tests (HST3 and HST4) in an environmental chamber (40°C; 40%RH). **INTERVENTION:** HST consisted of two 60-min bouts of treadmill exercise (~45% VO_2 max, 2%grade) with 10-min of hand cooling or passive rest following each bout. HA sessions consisted of 90-240 min of treadmill or stationary bike exercise at various intensities. **MAIN OUTCOME MEASURES:** Cooling rate, hand grip strength, hand volume, palm surface area, and physiological and perceptual measures associated with HA. A 3-way (acclimation x cooling x bout) repeated measures ANOVA with Fishers LSD *post hoc* ($\alpha < 0.05$) analyzed differences in cooling rate, rate of rise, and hand grip strength. Paired sample t-tests analyzed changes in physiological variables pre and post HA. Friedman analysses were used for all ordinal perceptual variables. Pearson product-moment correlations analyzed associations between cooling rates and both hand volume and palm surface area.

RESULTS: Compared to NHA, once HA, participants experienced a lower rectal temperature (39.21±0.18°C vs. 38.66±0.15°C, $p \leq 0.05$) and heart rate (158±5bpm vs.140±5bpm, $p < 0.001$) as well as higher sweat rate (1.25±0.08L/hr vs. 1.60±0.09L/hr, $p < 0.001$), confirming acclimation. When NHA, hand cooling (0.020±0.003°C/min) had greater cooling rate compared to no cooling (0.013±0.003°C/min) (MD [95%CI], p value; 0.01°C [0.00,0.01], p=0.035). When HA, hand cooling (0.021±0.002°C/min) was not different than no cooling (0.025±0.002°C/min) (0.004°C [-0.003,0.011], p=0.216). Rectal temperature rate of rise when HA (0.019±0.001°C/min) was lower than when NHA (0.022±0.001°C/min) (0.003°C/min [0.001,0.005], p=0.009). Once HA, grip strength decreased more following hand cooling compared to following no cooling (-2.1±0.7kg vs. -0.1±0.7kg) (2.0kg [0.3,3.7], p=0.023). There were no relationships between cooling rates and both palm surface area and hand volume ($p > 0.05$). **CONCLUSIONS:** Hand cooling improved cooling rates, beyond that of passive rest, in NHA conditions, however, no benefits occurred after HA. Passive cooling after HA provided the greatest cooling rate.

Key Words: rectal temperature, cooling rate, grip strength, hyperthermia, peripheral cooling

CHAPTER I

REVIEW OF THE LITERATURE

I. PHYSIOLOGY OF THERMAL STRESS

Human physiological responses are greatly impacted by exercise in hot, humid environments. Thermal stress, induced from the combination of exercise in hot ambient conditions, can lead to detrimental and often life-threatening consequences on the body's thermoregulatory mechanisms. Adaptations due to the acquisition of heat acclimation result in protective mechanisms that aid in prevention of heat illnesses. These adaptations may improve the efficacy of sideline cooling modalities, such as hand cooling devices, designed to mitigate the rise in core body temperature during exercise, as well as improve performance. The effectiveness of these cooling modalities warrants further research, especially in the realm of heat acclimation.

A. Heat Balance

Our bodies strive to maintain a thermal homeostasis through balancing physiological mechanisms of heat gain and heat loss. If heat gain is greater than heat loss, the resulting net heat gain leads to a rise in body temperature. In opposition, if heat loss is greater than heat gain, the resulting net heat loss leads to a decrease in body temperature. The heat balance equation presented below mathematically demonstrates the relationships between the physiological mechanisms involved thermal homeostasis.[1]

$$S = M - (\pm \text{Work}) \pm E \pm R \pm C \pm K$$

S = Heat storage

M = Metabolic heat production

Work = External work performed

E = Evaporation

R = Radiation

C = Conduction

K = Convection

Positive heat storage values (S) indicate heat gain, while negative values indicate heat loss. Evaporation, radiation, conduction, and convection are heat exchange pathways that can be utilized to dissipate heat gained by metabolic heat production.[1] Evaporation of sweat occurs through the conversion of liquid to gas, requiring an input of heat from the skin. This change in state is the predominant form of heat loss at high ambient temperatures. Radiation requires the movement of heat via electromagnetic waves down the thermal gradient. Conduction, usually negligible during exercise heat-stress, is the movement of heat between objects in direct contact. Convection is the exchange of heat between an object and its surrounding liquid medium, such as air, water, or body fluids. In all these stated pathways, heat moves in the direction of high thermal gradient to low. The environmental conditions in which exercise occurs strongly impacts which heat exchange pathway(s) play a primary role in thermoregulation.[2]

B. Thermoregulation and the Brain

Temperature regulation is coordinated by neurons originating in the preoptic area of the anterior hypothalamus (POAH).[3] Neural pathways extend from this region of the hypothalamus to the brainstem, spinal cord, sympathetic ganglia, and ultimately coordinate effector responses. It is believed that the POAH has a set point temperature of 37°C.[4] If core temperature rises above this set point, heat-sensitive neurons are activated to initiate heat loss responses. Conversely, if core temperature falls below this set point, activation of cold-sensitive neurons initiates heat gain responses. Temperature-insensitive neurons act to modulate both the efferent and afferent responses of these cold and warm-sensitive neurons.

C. Cardiovascular Responses

Cardiovascular responses to exercise heat stress include a redistribution of blood from the core to the periphery. Constriction of blood vessels in the visceral region allows for a greater blood volume available to the skin and working muscles. This sympathetic cutaneous vasodilation response can increase blood flow to the skin by up to 8 liters per minute.[5] In two studies by Rowell et al.,[6,7] a 20% decrease in splanchnic blood flow occurred during low to moderate exercise in unacclimatized men. This redistribution of blood leads to an increase in cutaneous venous volume, thus reducing ventricular filling pressure, end-diastolic volume, and stroke volume.[5] This phenomenon, termed cardiovascular drift, is characterized by a decrease in stroke volume with a parallel increase in heart rate to maintain cardiac output.[5,7-11]

Cardiovascular drift is impacted by numerous factors including, but not limited to, environmental temperature, hydration status, exercise intensity, and cooling modalities. Exercise in hot environmental conditions has been shown to significantly increase cardiovascular drift as well as decrease maximum volume of oxygen uptake (VO_2 max) compared to exercise in cool environments.[12] While the precise mechanism is not known, it is speculated that a reduced stroke volume during exercise in hot environmental conditions is the driving force behind this phenomenon. In a classic study by Ekelund,[8] subjects in a euhydrated state showed no change in blood volume beyond the initial ten minutes of exercise and remained steady until exercise completion, demonstrating cardiovascular drift can occur without dehydration. (Figure 1A) While this may be the case, dehydration greatly exacerbates cardiovascular drift (Figure 1B) due to the reduction in blood volume, which in turn decreases stroke volume.[9] Hypovolemia alone can reduce stroke volume up to 7%, but when combined with hyperthermia, the synergistic effect equates to a 20-28% decline.[9,11,13,14] In this situation, stroke volume is insufficient to maintain

cardiac output. High exercise intensity combined with hyperthermia can also lead to a decrease in cardiac output of up to 2 liters per minute less than that in a thermoneutral environment.[11] Competition for blood flow occurs between skeletal muscles and skin, resulting in a sacrifice of skin blood flow in order to maintain muscle metabolism.[5] This decrease in blood flow to the periphery impairs heat loss mechanisms and increases core temperature. If cardiac output is not sufficient to maintain muscle metabolism, muscle blood flow is then sacrificed to maintain arterial pressure.[5]

The addition of cooling during exercise via fan airflow has been shown to diminish the decline in VO_2 max and mitigate cardiovascular drift.[15] Given that cooling mitigated these cardiovascular repercussions, perhaps it effect on stroke volume, and thus in turn, cardiovascular drift and VO_2 max, provides rationale. Cooling decreases body temperature and therefore reduces the need for redistribution of blood to the periphery. This helps to maintain central venous pressure, end diastolic volume, and consequently stroke volume. While this proposed mechanism is just that, a potential mechanistic rationale, other cooling modalities should be studied in this context to fully understand the cardiovascular benefits cooling can provide to exercise-heat stress.

Cellular Responses

Excessive hyperthermia during exercise is associated with a cascade of damaging reactions, starting at the cellular level. Splanchinc vasoconstriction, resulting in a decreased blood flow, can result in ischemia and mild endotoxemia of the gastrointestinal region.[16,17] Proposed mechanisms for the breakdown in barrier function of the intestines include the disruption of normal epithelial mucosa integrity and/or failure of epithelial tight junctions.[18,19]

This endotoxemia plays a pivotal role in the pathophysiology of systemic inflammatory response syndrome (SIRS) seen in individuals with severe heat illnesses such as exertional heat stroke (EHS).[20-22] Death from EHS caused by multi-organ system failure is likely due to the combined effects of cytotoxicity, coagulopathies, and SIRS.[20] (See Figure 2) Cytokines, or immune modulators, involved in SIRS are thought to elicit time and tissue specific protective actions that aid in the resolution of this inflammation.[21,23,24] An understanding of these cellular responses gives deeper insight into the severity of this potentially life threatening condition, as well as clear rationale for the critical need of knowledge on ways for prevention. The reduction of body temperature through preventative strategies (e.g. physiological adaptations of heat acclimation and body cooling) helps to prevent this damaging cellular cascade.

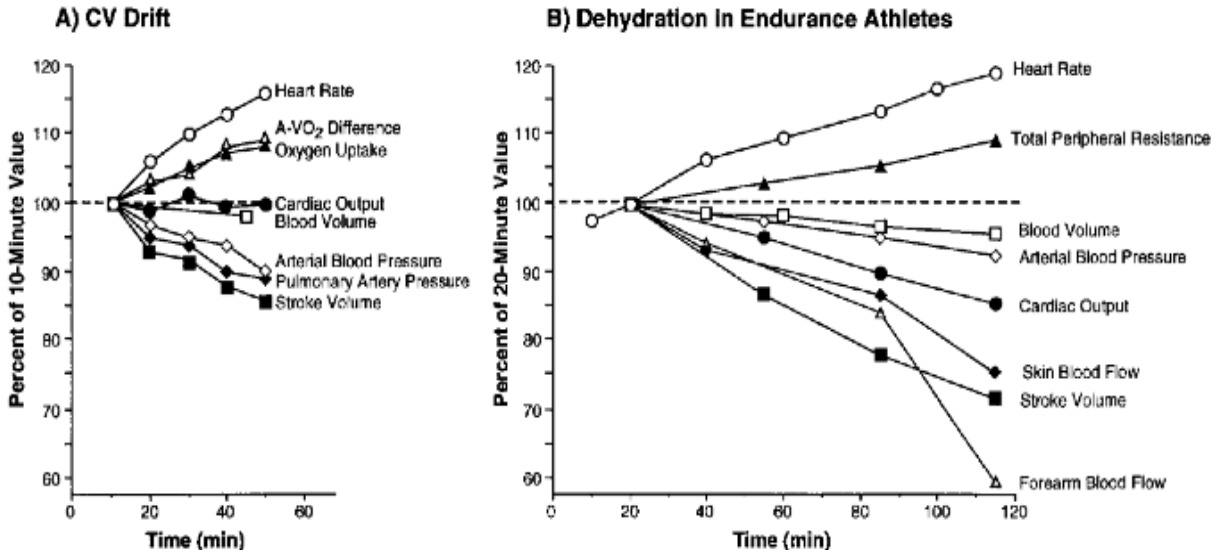

Figure 1. A) Cardiovascular changes in a euhydrated state. B) Effects of dehydration and hyperthermia during 120 minutes of cycling exercise at 62-65% VO2 max and 35°C. Source: Ekelund LG. Cardiovascular drift during prolonged exercise: new perspectives. *Exerc. Sport Sci Rev.* 2001;29(2):88-92.

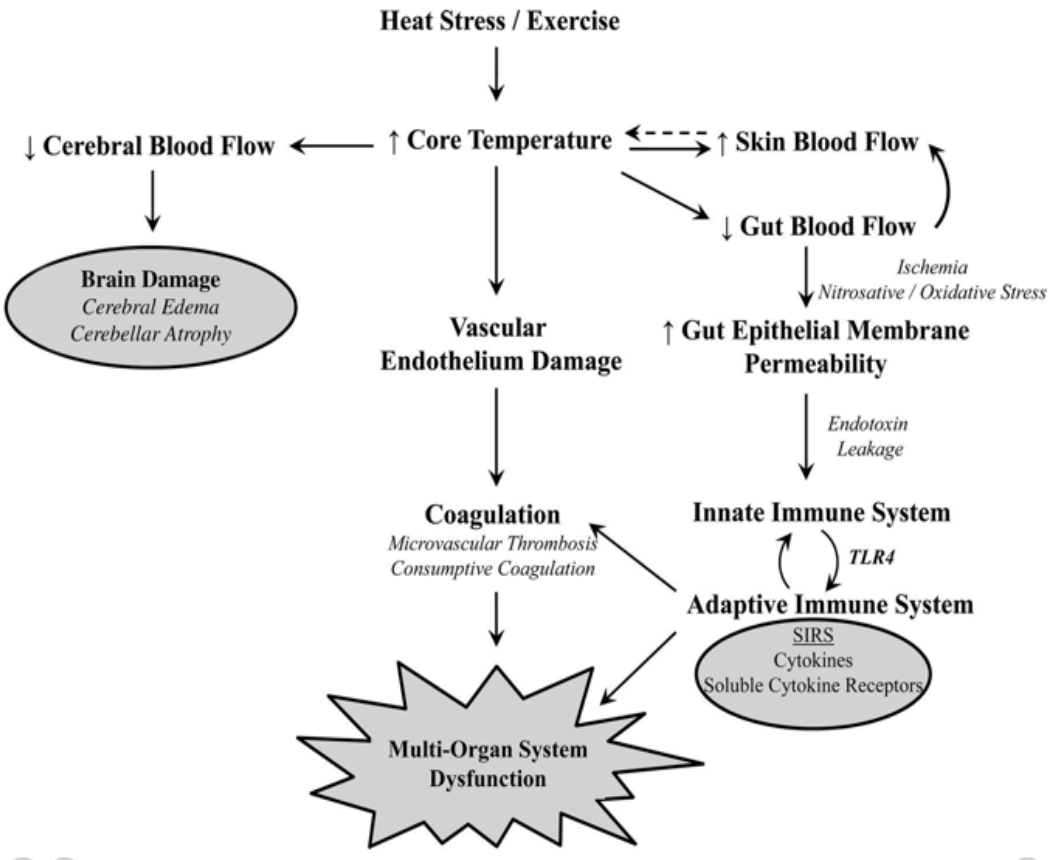

Figure 2. Summary of heat stroke pathophysiological changes culminating in multi-organ system dysfunction and death. Source: Lim CL, Mackinnon LT. The roles of exercise-induced immune system disturbances in the pathology of heat stroke: the dual pathway model of heat stroke. *Sports Med.* 2006;36:39-64

E. Exertional Heat Illnesses

Four types of exertional heat illnesses (EHI) can occur during prolonged intense activity in the heat. These maladies include exercise-associated muscle cramps, heat syncope, heat exhaustion, and exertional heatstroke.[25] Heat exhaustion and exertional heatstroke (EHS) will be discussed further given the severity of these heat illnesses. These two conditions involve elevations in core temperature (36^{o}C-40^{o}C and >40.5^{o}C, respectively).[25] Both occur most frequently in hot and humid environments. Heat exhaustion is defined as the inability to continue exercise in combination with heavy sweating, dehydration, sodium loss, and energy

depletion. EHS is characterized by a core temperature above 40.5°C (105°F) and central nervous system dysfunction. EHS is a medical emergency, and if not treated immediately and aggressively, multisystem organ failure can lead to permanent disability or death. In order to help prevent this serious EHI, the body can undergo physiological adaptations in a process of heat acclimation.

II. HEAT ACCLIMATION

Heat acclimation (HA) is the improved ability to exercise in a hot environment due to physiological adaptations advantageous for heat removal. This protective mechanism is achieved through a process of repeated exposures to exercise-heat stress in an artificially controlled environment. While heat acclimation and acclimatization have been used synonymously, the terms differ in that acclimatization occurs in a natural environment. It is well established that HA results in numerous physiological adaptations shown to help individuals cope with exercise in the heat[26] as well as reduce the incidence of exertional heat illness[25,28] and improve performance. These beneficial adaptations include cardiovascular changes, sweat responses, and overall decrease in core temperature.

A. Physiological Adaptations

The cardiovascular adaptations resulting from HA include: hypervolemia induced plasma volume expansion, increased stroke volume, decreased heart rate, increased cardiac output, and enhanced vasodilatory properties of cutaneous vasculature during exercise. Hypervolemia is an increase in circulating blood volume greater than normal. The increase in intravascular protein concentration with a constant plasma protein concentration causes a hypervolemic response, thus increasing plasma volume.[29-31] Haemodilution, due to the increase in plasma volume, causes a

shift of protein from the interstitial to the intravascular space in order to maintain pressure within the capillaries, thus providing evidence that oncotic factors are a driving force in the maintenance and regulation of plasma volume.[31-35] Plasma volume change during exercise, calculated using blood hematocrit and hemoglobin values,[36] increases in the range of +3 to +27%.[37] This leads to many additional cardiovascular changes during exercise, ultimately reducing cardiovascular strain. These adaptations include an increase in stroke volume and cardiac output with a decrease in heart rate. The increase in venous return to the heart increases ventricular filling pressure, thus expanding stroke volume by 10-20%,[38-42] while reducing maximal heart rate by 15-25%.[43] Figure 3B[44] depicts the decline in heart rate over successive days of HA. As a result of the increased stroke volume and decreased heart rate, total cardiac output is increased. This adaptation results in a greater blood volume available for cutaneous perfusion, thus widening the core-to-skin thermal gradient and enhancing skin blood flow.[44,45] The physiological mechanism driving augmentation of skin blood flow due to HA remains inconclusive.[46-50] One theory suggests a central mechanism lowers the internal temperature threshold for cutaneous vasodilation with no significant change in the slope of this relationship.[47] Conversely, some studies have shown an increase in the slope of the relationship between vascular conductance and internal temperature.[46,49] It remains unclear whether the mechanism is centrally driven or if structural changes occur in the peripheral vasculature; however, in all studies, skin blood flow is increased as a result of a higher internal temperature during exercise-heat stress following a period of HA.

Additional adaptations of HA include increased sweat rate, improved sweat sensitivity, and decreased sodium chloride in sweat and urine; however, these are highly dependent upon environmental conditions[51,52] and diet.[37] The increase in sweat rate during HA can take up to 14

days to reach a plateau as illustrated in Figure 3A[44] and Figure 4.[53] Similar to the previous discussion in regards to blood flow, it is inconclusive as to whether the increase in sweating capacity is largely due to an increase in the secretory activity of the sweat glands[54-61] or an increase in stimulation from the central nervous system.[47,59 62-64] Currently there is more literature supporting the peripheral rather than centrally mediated mechanism; however, the earlier onset of sweating and occurrence at lower body temperatures due to HA appears to be driven by central nervous control of the thermoregulatory system.[62,65]

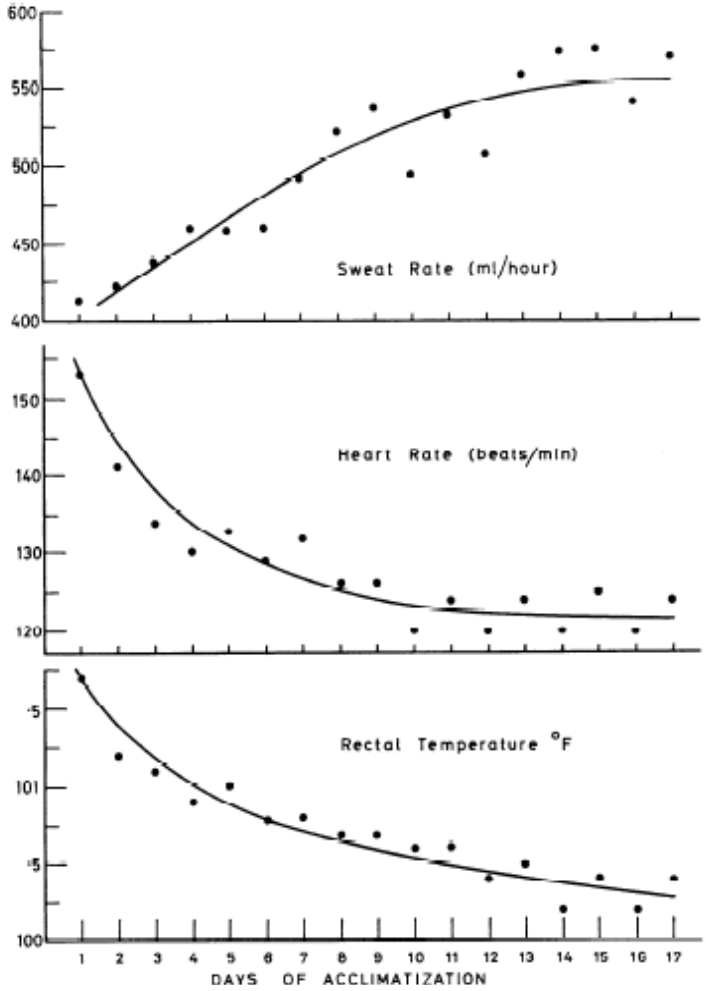

Figure 3. The relationship of A) sweat rate B) heart rate and C) rectal temperature over the course of 17 days of heat acclimatization. Source: Wyndham CH, Benade AJA, Williams CG, et al. Changes in central circulation and body fluid spaces during acclimation to the heat. *J Appl Physiol.* 1968;25:586-593.

Due to the improvements in cardiovascular function and sweat response from HA, an overall attenuation of core body temperature during exercise in the heat is achieved. (See Figure 3C) For example, the importance in plasma volume expansion rather than blood volume expansion is shown by two studies by Fortney et al.[66,67] In the first study,[66] an increase of 10% in total blood volume by reinfusion of subjects own blood was shown to have no effect on core temperature during a 30-minute cycle exercise at 60% VO_2 max. In contrast, under the same experimental conditions, an 8% increase in blood volume by infusion of isotonic serum albumin did result in a significantly lower core temperature.[67] Albumin maintains intravascular onconic pressure, and therefore an increase contributes to the interstitial-to-intravascular plasma fluid shift as previously described. These physiological changes indicate that an absolute increase in total blood volume does not reduce core temperature; however, an increase in plasma volume does, in fact, contribute to a reduced core temperature. In addition to decreased attenuation of core body temperature during exercise, acclimation in humid-heat as shown to reduce resting core temperature.[68] A decrease in core temperature is a one of the most critical adaptations of HA.

Table I. 'Plateau days' of physiological adaptations (the point at which approximately 95% of the adaptation occurs) during heat acclimatisation (from Armstrong & Dziados 1986)

Adaptation	Days of heat acclimatisation													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Heart rate decrease			—	—	—	—								
Plasma volume expansion			—	—	—	—								
Rectal temperature decrease					—	—	—	—						
Perceived exertion decrease			—	—	—	—								
Sweat Na^+ and Cl^- concentration decrease[a]					—	—	—	—	—	—				
Sweat rate increase								—	—	—	—	—	—	—
Renal Na^+ and Cl^- concentration decrease			—	—	—	—	—	—						

a While consuming a low NaCl diet.

Figure 4. Course of days in which 95% of the physiological adaptations of heat acclimation occur. Source: Armstrong LE, Maresh CM. The induction and decay of heat acclimatization in trained athletes. *Sports Med.* 1991;12(5)302-312.

B. Rate and Efficacy

Different systems of the human body adapt at different rates, thus some HA adaptations occur before others. The progression of adaptations occurs in stages as seen in Figure 4.[53] It can take up to 14 days for full HA in all body systems; however, highly trained athletes have shown significantly enhanced cardiovascular and thermoregulatory benefits in short-term HA of just 5 days.[69] A high core body temperature has been suggested as the key determinant in adaptations to HA, therefore high-intensity intermittent exercise producing a great thermal strain may induce HA adaptations in less time than continuous moderate exercise.[70,71,72]

Individual characteristics such as age, aerobic fitness, body fat percentage, and gender have been suggested to influence rate and/or efficacy of HA. Studies have shown older men have higher heart rates, mean skin temperature, and core temperature, along with lower sweat rates during work-heat stress compared to younger men both pre- and post acclimation.[73,74] This suggests age hinders HA efficacy; however, in studies where subjects were matched for level of aerobic fitness, surface area-to-body mass ratio, and body fat percentage, physiological heat strain during acute heat stress and HA was the same or improved in middle-aged men and women compared to those who were younger.[75-79] These findings support aerobic fitness and morphological factors as having a greater importance than aging on thermoregulatory performance during work in the heat. Given that the efficacy of HA is dependent on the fitness status of the individual, the higher the background adaptation, the lower the adaptation response.[80,81] One investigation found that individuals with a VO_2 max of 65ml/kg/min required only 4 days to reach a stable plateau of rectal temperature and heart rate, while those with a VO_2 max of 50 and 40ml/kg/min required an average of 6 to 8 days respectively to reach the same plateau.[82] Vasodilation and sweating thresholds appear to be higher in women, implying less

skin blood flow and lower sweat rates than men at the same skin and core temperatures.[47, 83] Women, however, have a smaller blood volume; so relatively speaking any gender difference will be minute. Few studies have found significant gender-related heat tolerance differences.[84,85]

C. Practical Application

High intensity exercise in the heat can induce heat illnesses in any athlete, so HA is critical in reducing the signs and symptoms, as well as indices of most forms of heat illness.[86] It has been shown that HA individuals are less likely to have heat cramps,[86,87] and the cardiovascular adaptations of HA, such as increased plasma volume and cutaneous blood flow, result in a decline of heat syncope on the first 2-5 days of heat exposure.[88] Signs and symptoms of heat exhaustion have been shown to decrease during 8 days of HA.[89] While HA induces many physiological changes that very likely provide protection from heat stroke, it should be noted that there is no clear relationship between HA and heat stroke.[90]

HA may improve exercise performance through physiological changes including but not limited to: reduced oxygen uptake at a given power output,[91,92] muscle glycogen sparing,[92,93] reduced blood lactate at a given power output,[92] increased skeletal muscle force generation,[94] improved myocardial efficiency,[95] and increased ventricular compliance.[96] Two studies that examined running economy in HA individuals found a 3%[91] and 4%[71] increase in submaximal VO_2 during treadmill exercise at controlled speeds. This is important to endurance athletes since it means less oxygen is utilized for a given amount of work. Additionally Lorenzo et al.,[97] showed improvements in HA individuals during exercise performance in both hot and cool conditions. Specifically, a 5% increase in VO_2 max, 6% increase in total work completed during a time trial, and 5% increase in power output at lactate threshold in a cool environment was observed in HA individuals. While repeating these measures in a hot environment, even greater

performance benefits of an 8% increase in VO_2 max, 8% increase in total work completed during the time trial, and 5% increase in power output at lactate threshold were seen. These results are illustrated in Figure 3. In conclusion, from a clinical applications perspective, HA not only keeps athletes safe by mitigating signs, symptoms, and indices of heat illnesses but also improves exercise performance.

III. COOLING MODALITIES

A. Cooling Rates

When performing exercise in hot environmental conditions, it is critical that one's core temperature does not become dangerously elevated. In order to prevent and/or treat a high core body temperature, different cooling modalities can be utilized. The most important characteristic of cooling modalities is their cooling rate or ability to remove heat from the body over a given unit of time. Studies examining multiple types of cooling methods have shown variance in ability to decrease core temperature depending on multiple factors, including both exercise and cooling mode and duration, as well as exercise intensity. [98,99]

When treating a serious heat illness such as exertional heat stroke (EHS), the main criterion for survival is the length of time the core temperature exceeds the critical threshold for cell damage.[100,101] For this reason, rapid cooling, aimed at lowering body temperature as quickly as possible, is the most important element in the treatment of EHS. The cooling modality used must be immediately accessible and able to reduce body temperature to below 40°C in less than 30 minutes.[102] If cooling begins immediately, body temperature cooling rate should exceed 0.1-0.2°C•min^{-1} and if delayed, values should be no less than 0.15°C•min^{-1}.[103] Cold water immersion (CWI) has shown to provide the fastest cooling rates in treating

hyperthermia.[103,104] (See Figure 5) Due to the physical characteristics of water, CWI has the ability to cool at rates 0.15°C•min^{-1} or greater, far superior than any other cooling method.[103] CWI has been termed the "gold standard" for treatment of EHS and should be the preferred modality when treating serious heat illnesses.[103]

For cooling modalities used to aid in prevention of hyperthermia, a slightly lower cooling rate may be acceptable. These modalities are not used for treatment in life threatening situations but instead for attenuating rise in core body temperature to prevent heat illnesses and improve performance. Some common current sideline cooling devices are cooling collars, cooling towels, cooling vests, cooling jackets, and hand cooling refrigerant packs. The research on these cooling modalities is highly inconclusive.[105-114] In a field study by DeMartini et al.,[98] multiple cooling methods were examined for effectiveness on core body temperature, HR, and perceptual responses. Exercise occurred in warm outdoor conditions, intensity was self-paced, and cooling modalities were applied after 45-60 minute bouts. The drop in core temperature over time for all devices tested can be seen in Figure 6. These devices include cold water immersion (CWI), control (SUN), shade, fan, Emergency Cold Contaminant System™ (ECCS), Rehab Hood™ (HOOD), GameReady™ Vest (GRV), Nike Ice Vest™ (NIV), ice buckets (IB), and ice towels (IT). Compared to the control group (1.50°F drop in 17 min), CWI, NIV, and IB all had faster cooling rates of 1.50°F in 14, 16, and 12 minutes respectively. The remainder of the cooling modalities had slower cooling rates compared to the control group. In should be noted that a limitation of this study were the core body temperatures of subjects prior to cooling with an average temperature of 38.73°C. This may have limited these modalities' capability for cooling. Further studies have looked at cooling rates using hand and/or forearm immersion during and

after exercise.[115-117] These studies showed a variety of positive and negative results in cooling rate and exercise performance indicating the need for further research.

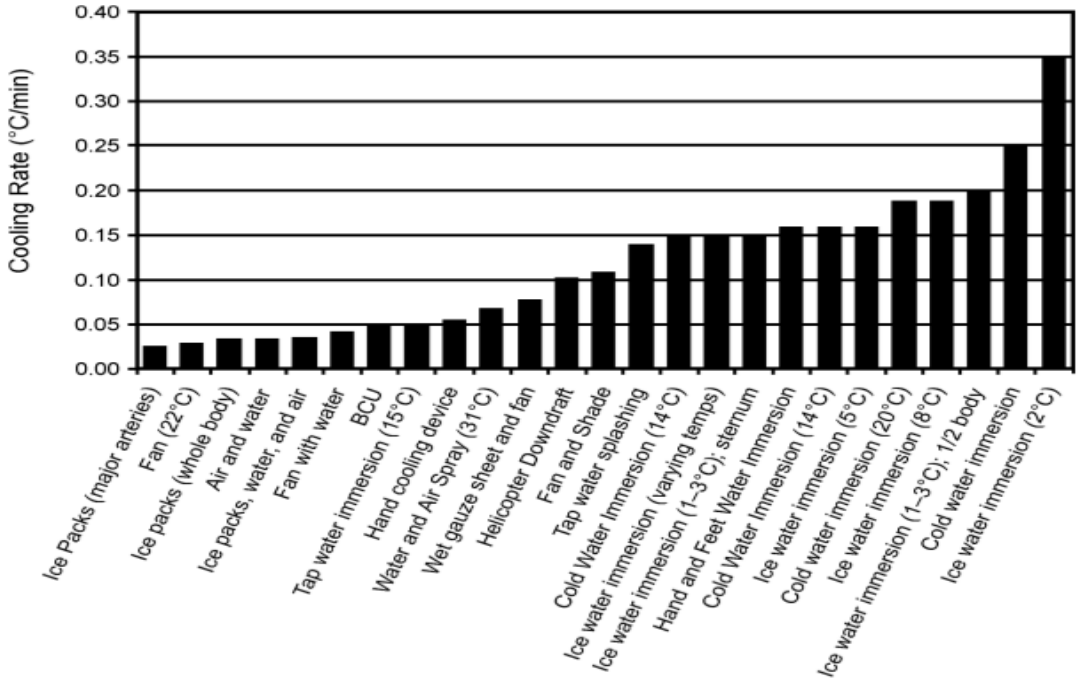

Figure 5. Mean cooling rates from case reports and reviewed articles. Mean cooling rates defined as unacceptable: < 0.078°C•min^{-1}; acceptable: 0.078°C-0.154°C•min^{-1}; ideal: ≥ 0.155°C•min^{-1}. Source: Casa DJ, McDermott BP, Lee EC, et al. Cold water immersion: the gold standard for exertional heatstroke treatment. *Exerc Sport Sci Rev.* 2007;35(3): 141-149.

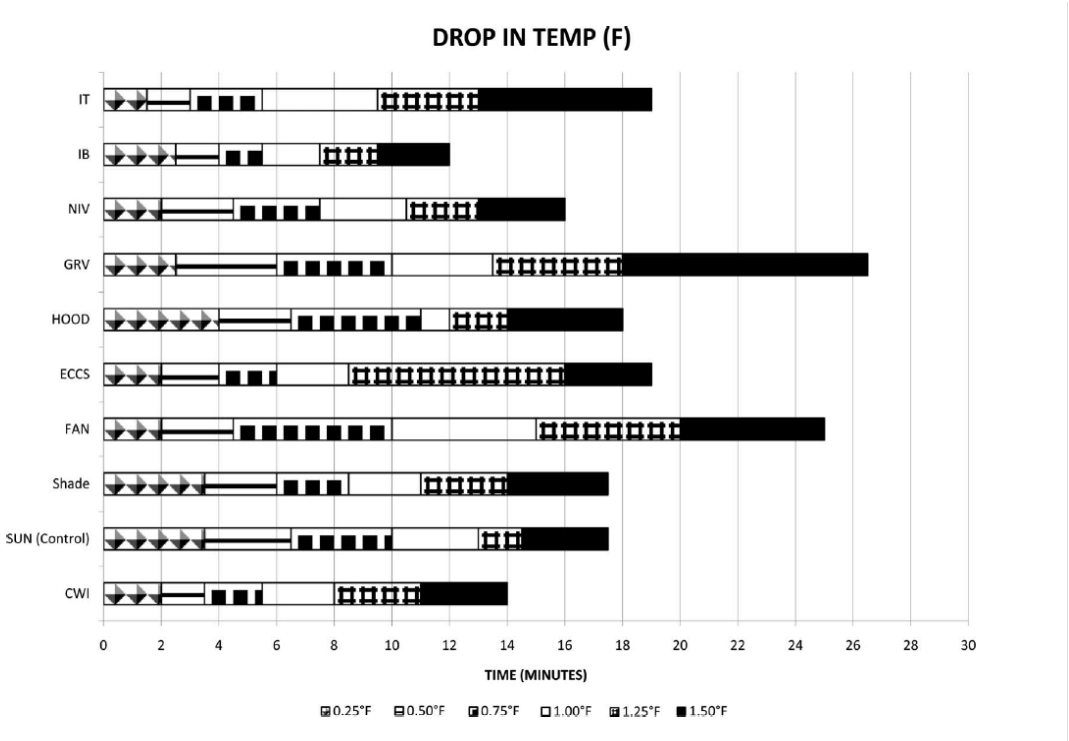

Figure 6. Drops in rectal temperature in 0.25°F increments over time for each modality. 0-minute time point= immediately before cooling; 10-minute time point= end of cooling phase; 30-minute time point = end of monitoring phase. Source: DeMartini JK, Ranalli GF, Casa DJ, et al. Comparison of body cooling methods on physiological and perceptual measures of mildly hyperthermic athletes. *J Strength Cond Res.* 2011;25(8):2065-2074.

B. Physiology of Hand Cooling

Humans have two types of skin, non-glabrous and glabrous, which differ in the presence or absence of hair follicles respectively. Glabrous skin, found in humans on the soles of the feet, palms of the hands, and regions of the face, contain heat transfer units which consist of dense subcutaneous vascular networks referred to as retina venosa and arteriovenous anastomeses (AVAs).[118] AVAs are unique vascular structures which shunt blood to the retina venosa, bypassing nutritive capillary beds. The retina venosa is a network of veins located in the dermal layer of skin below the papillary layer. In a study by Manelli et al.,[119] electron microscopy analysis (SEM) was used to obtain 3D images of glabrous skin regions. The images revealed a

hypodermal layer densely packed with sweat glands, AVAs, and glomerular-shaped vessels. These vessels are large in diameter allowing for the ease of movement and flow and provide a low-resistance pathway.[118,119] Blood flow through these glabrous regions is under vasomotor control and can be regulated via smooth muscle depending on thermoregulatory requirements.[120-122] Central nervous control of vasomotor activity in the AVAs has been shown to be related to central core temperature.[123-125] During heat stress, dilation of the AVAs increases blood flow to the glabrous skin regions in magnitudes greater than that of non-glabrous regions thus promoting heat exchange between the body's core and the surrounding environment.[126-128] These structures are able to accommodate an abundance of blood flow when dilated, and therefore maximize heat exchange.[118,119,128]

It has been documented that internal body heat can be extracted through the extremities.[127-129] In addition, the application of sub-atmospheric pressure on glabrous skin has been shown to largely increase blood flow to the fingers and toes.[130,131] In a study by Grahn et al.,[132] results showed a 6 times greater transfer of heat between glabrous skin and the body's core when sub-atmospheric pressure was applied to the forearm and hand. In a study by Heller and Grahn,[126] the addition of a mild vacuum to a hand cooling device increased heat loss from glabrous skin by an additional 33%. These studies conclude that the rate of heat transfer between glabrous skin and an outside source is directly related to the amount of blood flow and temperature gradient between the blood and external environment. The combined increase in blood flow to the hand via sub-atmospheric pressure and application of a cool external environment work together in decreasing internal body heat. This is the theory behind the CoreControl™ device by AVAcore Technologies Inc.

C. CoreControl™

This portable hand-cooling device is designed to enhance heat extraction through glabrous skin regions as described above. The exercising individual places their hand into a pod with a flexible airtight seal extending over the forearm. Within the pod, the palm faces down, resting on a perfusion pad. Temperature-controlled water of 16°C circulates underneath the perfusion pad while simultaneously, a vacuum source and pressure sensors create a slight sub-atmospheric pressure of ~40mmHg. This pressure enables more blood flow into the hand, thus allowing for increased heat dissipation via vasodilatation of the subcutaneous veins.[125] The cooled blood from the palm then returns to central vasculature via venous return and circulates throughout the body to working muscles. The extraction of heat from the blood in the palm allows for a greater capacity of the circulating blood to extract heat from the working muscles. In theory, this should reduce the amount of heat accumulation in active muscles and aid in thermoregulation.

This CoreControl™ device has been used in a number of studies examining its effects on core temperature, exercise duration, and volume of work during resistance training. Results have varied tremendously with very few performance benefits or significant cooling rates shown using this device. For example, one study analyzed the difference of hand cooling with and without sub-atmospheric pressure as well as with one or two hands.[133] Subjects cooled for 60 minutes following a 45-minute bout of exercise in military wear. Hand cooling on both hands with sub-atmospheric pressure showed a greater cooling rate (1.3°C□60min^{-1}) compared to one hand with sub-atmospheric pressure (1.0°C□60min^{-1}). If this data were to be converted to °C□min^{-1}, as in most existing literature, this device would have a cooling rate of approximately 0.02°C□min^{-1}

and $0.01 \cdot 1 min^{-1}$ respectively. The chart in Table 1 shows some of the current studies using the CoreControl™ device and their outcomes.

Table 1. Current CoreControl™ hand cooling studies and their results.

Author	n=	Exercise Type	Environmental Conditions	When cooled?	Cooling duration	Temperature Device	Cooling Rate
Hostler[134]	18	Walking	35.1 ± 2.7°C	Between exercise bouts	20 min	Gastrointestinal	0.040°C/min
Grahn et al[135]	17	Walking	41.5°C 20-30%RH	After exercise	60 min	Esophageal	0.02°C/min
Grahn et al[135]	18	Walking	40°C 20-30%RH	During exercise	20-45 min	Esophageal	0.001°C/min
Grahn et al[135]	8	Walking	41.5°C 20-35%RH	After exercise	3 min	Esophageal	0.6°C mean difference
Amorium et al[115]	10	Walking	42.0°C 30%RH	After exercise	41 min	Rectal	0.02°C/min
Zhang et al[136]	8	Walking	33.7°C 40-45%RH	After exercise	40 min	Rectal	0.02°C/min
Kuennen et al[138]	10	Walking	42.2°C 35.5%RH	After exercise	50 min	Esophageal	0.015°C/min
Grahn[133] (one hand)	17	Walking	41.5°C 20-35%	After exercise	60 min	Esophageal	0.02°C/min
Grahn[133] (two hands)	17	Walking	41.5°C 20-35%	After exercise	60 min	Esophageal	0.02°C/min
Grahn[133] (no pressure)	17	Walking	41.5°C 20-35%	After exercise	60 min	Esophageal	0.013°C/min

IV. Gaps in the Literature

Currently scientific literature regarding the CoreControl™ hand cooling device efficacy remains inconclusive. More research is needed to determine its effectiveness as a cooling modality for the prevention of hyperthermia and enhancement of athletic performance. Specifically, cooling rates of this device following HA have not been studied. It is possible that lower core body temperatures observed in heat acclimated individuals following exercise, as

compared to when not heat acclimated, results in a smaller heat gradient and thus potential for diminished cooling rates. In addition, the effect of hand size on cooling rate of the CoreControl™ device is unknown. It is thought that a larger hand will have a greater cooling rate due to the increased surface area available for cooling. Those with larger hand sizes may benefit more from this cooling modality. Finally, the effect of hand cooling on grip strength is also unknown. It has been shown in two Kwon articles[138,139] that hand cooling increases bench press reps performed, perhaps due to a cooler, more optimal muscle temperature able to produce a greater work output. If this same optimal temperature is achieved prior to a hand grip strength test, a larger force may result. In order to better understand the full potential for hand cooling to aid in prevention of heat illnesses, there needs to be further investigation on its cooling capabilities in these different circumstances.

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CHAPTER II

INTRODUCTION

Strenuous physical activity in hot, humid environments places individuals at great risk for heat-related illnesses such as heat exhaustion and exertional heat stroke. When the body's heat gain exceeds its heat loss, thermal homeostasis is disrupted and an increase in core body temperature results.[1] This increase in core body temperature can have life threatening consequences if not treated appropriately. Protective strategies, such as heat acclimation (HA) or body cooling during exercise, can be utilized for prevention of these often-fatal conditions.

Cardiovascular changes during exercise heat stress include visceral vasoconstriction in conjunction with cutaneous vasodilation. This physiological response redistributes blood from the core to the periphery, shunting blood to the skin's surface to enhance the sweating mechanism. This process ultimately reduces stroke volume while increasing heart rate to maintain cardiac output.[2] This phenomenon is termed cardiovascular drift.[2-7] When high intensity exercise is combined with hyperthermia, an overall decrease in cardiac output may result in a competition for limited blood flow. Ultimately, blood flow to the skin and muscles is sacrificed in order to maintain arterial pressure.[2] This reduced blood flow, specifically to the splanchnic region, may result in ischemia and mild endotoxemia.[8,9] Endotoxemia is paramount to the pathophysiology of systemic inflammatory response syndrome, seen in individuals with exertional heat stroke.[10-12] Physiological adaptations associated with HA are crucial in preventing this whole-body organ failure.

HA is a period of repeated exposures to exercise heat stress during which the body undergoes physiological changes advantageous for heat removal. HA has been shown to reduce incidences of heat illnesses,[13,14] help individuals cope with exercise in the heat,[15] and improve performance.[16,17,18] Occurring over a 10-14 day period,[19] these physiological adaptations include,

but are not limited to: increased plasma volume, increased stroke volume, decreased heart rate, increased sweat rate, earlier onset of sweat during exercise, and decreased concentration of sodium chloride in sweat. Due to these cardiovascular and sweat response changes, an attenuation of core body temperature during exercise is achieved. Additionally, resting core body temperature is also reduced following humid-HA.[20] The overall decrease in core body temperature is arguably the most crucial adaptation of HA.

Body cooling during exercise in the heat is another strategy for mitigating rise in core body temperature. Some current cooling modalities used to aid in the prevention, not treatment, of hyperthermia include cooling collars, vests, and jackets, forearm and hand immersion, and hand cooling devices.[22-30] Hand cooling devices (e.g. CoreControl^{TM}) are unique in that the surface area they cool, the palms of the hands, contain vascular networks referred to as arteriovenous anastomeses.[31] These vessels are large in diameter and bypass capillary beds, allowing for ease of movement. During heat stress, these vessels dilate thus accommodating an abundance of blood flow to maximize heat exchange with the surrounding environment.[32-34]

The CoreControl^{TM}, in particular, is a portable hand cooling device made of a plastic pod in which the individual places their hand. An airtight seal is created surrounding the forearm, cool water (16°C) is circulated beneath the palm, and a slight sub-atmospheric pressure (~40mmHg) is maintained by a vacuum source. Of the limited studies examining this device, its effects on core temperature, exercise duration, and work volume during resistance training remain varied. Results often demonstrate less than optimal cooling rates (e.g. 0.020°C/min-0.040°C/min);[35-39] however, when used between bench press sets, work output has been shown to improve.[42-43] More research is needed to fully understand this devices' effectiveness. Specifically, the efficacy of the CoreControl^{TM} once individuals are HA has not been studied.

Both the physiological adaptations of HA and the hand cooling device independently aid in heat dissipation; however, the combined effects, whether synergistic or invariable, have not been explored. Therefore, the primary purpose of this study was to compare the separate and combined effects of HA and hand cooling on cooling rate, during and after exercise in the heat. Our secondary aims were to determine the influence of hand cooling on grip strength, as well as examine the relationship between cooling rate on hand volume and palm surface area. We hypothesized that hand cooling and HA together would provide the greatest cooling rates compared to HA or hand cooling alone; additionally, hand cooling would improve grip strength, and cooling rates would have a positive correlation with hand volume and palm surface area.

CHAPTER III

METHODS

Participants

Healthy, non-HA (NHA), recreationally active males were enrolled as participants in this research study. This laboratory study was conducted in accordance with the University of Connecticut International Review Board, and participants enrolled completed an informed consent. Inclusion criteria were as follows: (a) 18-35 year old English speaking males of any ethnicity or income level, (b) maximum volume of oxygen consumption (VO_2 max) greater than 45 ml/kg/min, (c) no chronic health problems, (d) no previous history of exertional heat stroke within the past 3 years, (e) no history of cardiovascular, metabolic, or respiratory disease, (f) no current musculoskeletal injury that limits physical activity. Participants completed a self-administered, physician approved, medical history questionnaire to ensure they met the above criteria. Female participants were not enrolled due to the influence of menstrual cycle on core body temperature and sweat gland function.[44.45]

Protocol

Participants attended sixteen total sessions: one baseline session that included aVO_2 max test, two pre-HA heat stress tests (HST1 and HST2), ten HA exercise sessions, two post-HA heat stress tests (HST3 and HST4), and a final VO_2 max test. Baseline and VO_2 max tests occurred in a thermoneutral environment. All other testing occurred in a temperature-controlled environmental chamber (4284-2L-W, Minus-Eleven, Weymouth, MA). Ambient temperature and humidity were maintained at 40°C, 40%RH as measured by a handheld wet blub globe temperature meter (4400 Heat Stress Tracker Kestrel, Birmingham, MI). This study was

conducted between the months of October and February to ensure participants were not HA. Figure 7 contains a visual representation of the study timeline.

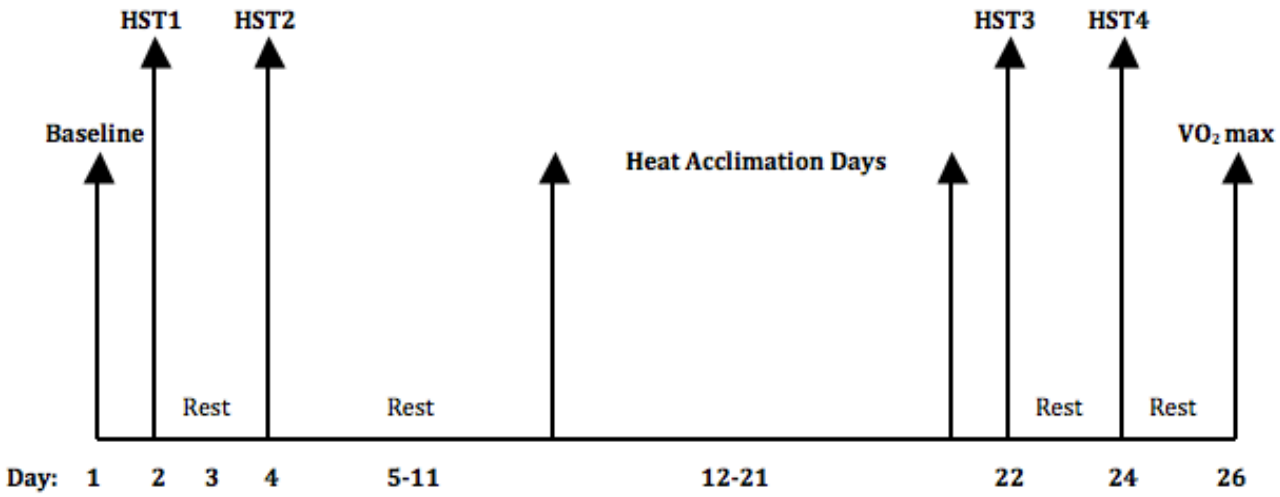


Figure 7. Study timeline depicting all testing sessions. On days 3, 5-11, 23, and 26 no testing occurred in order to prevent previous testing session from influencing subsequent days. HST=heat stress test.

Baseline Measures

Demographics: Participants began by completing an exercise history questionnaire in order to quantify weekly aerobic activity. Height was measured using a stadiometer with participants shoes removed. Baseline body mass was measured on a scale (T51P, Ohaus, Pine Brook, NJ) with participants shoes removed and minimal clothing worn. Body surface area was calculated using the DuBois equation.[46] Body fat percentage was measured using a Lange skinfold caliper (Lange, BetaTechnology Incorporated, Cambridge, MD). A minimum of two measurements occurred at three different sites (chest, abdomen, and thigh), with a third measurement taken when there was a larger than two numerical discrepancy.[47]

Hand size: Hand size was quantified by using two measures: hand volume and palmar surface area. Hand volume was measured using water displacement (Figure 8). The participant submerged each hand into a clear cylinder filled to the brim with water. The amount of water displaced from the cylinder denoted the volume of the hand. Palmar surface area was measured by tracing the hand as it lies palm side down on a sheet of paper. Hand length and span of the palm were used to calculate surface area.[48]

Figure 8. Hand volume measurement by water displacement.

Grip strength: Grip strength was measured using a hand grip dynamometer (Jamar, Creative Health Products Inc., Ann Arbor, MI). Participants were seated with elbow flexed to 90°, resting forearm on a table, and wrist in a neutral position off the table (Figure 9). Participants applied maximal gripping pressure to the dynamometer a total of 3 times for each hand, separated by 30 seconds rest, to determine average grip strength of each hand.[49]

Figure 9. Grip strength measurement utilizing a hand dynamometer.

VO_2 max test: Participants performed a baseline graded exercise test on a treadmill (956I, Precor, Woodinville, WA) utilizing a metabolic cart (TrueOne® 2400 Metabolic Measurement System, Parvo Medics, Sandy UT). After a 5-minute, self-administered warm-up, the ramping protocol

consisted of participants walking at a 2% grade. Every two minutes, treadmill speed was increased until voluntary exhaustion. VO_2 max was confirmed if participants met 3 out of the 4 following criteria: 1. Within 15% of age predicted maximum heart rate (220-age) 2. Rating of perceived exertion greater than 17. 3. Plateau in VO_2 values during the last two stages of exercise. 4. RER greater than 1.0. VO_2 max was used to ensure participants met inclusion criteria and to determine relative intensities for HST. This same protocol was used for the final VO_2 max test.

Hand Cooling Device: The CoreControl™ portable hand cooling device consists of a hard plastic pod with a flexible airtight seal extending over the forearm (Figure 10). Within the pod, one's palm faces down onto a perfusion pad in which cold circulating water (16°C) flows. Once the airtight seal is created, a sub-atmospheric pressure (~40mmHg) is maintained through a vacuum pump.

Figure 10. CoreControl™ hand cooling device.

Heat Stress Tests

Participants were instructed to drink 500mL water the night before and 250mL the morning of each visit to ensure proper hydration. Upon arrival to the lab wearing a T-shirt and shorts, a urine sample was collected in a clean urine cup to determine urine specific gravity using a refractometer (A300CL, Atago, Bellevue, WA) and urine color.[50] Euhydration was defined as urine specific gravity ≤ 1.020. If participants were determined to be hypohydrated, they were given a weighed water bottle (CI4000, Jennings, Phoenix, AZ) containing approximately 500mL

water to consume prior to the start of exercise. Participants privately inserted a flexible rectal thermometer (401, Measurement Specialties, Beavercreek, OH) 10cm past the anal sphincter in order to assess rectal temperature (4600 Thermometer, Measurement Specialties, Beavercreek, OH). Nude body mass was obtained in a private location with the participants alone on a scale behind a closed door, with the researcher reading the display outside of the room. Change in body mass from pre and post HST was used to determine sweat rate during the session. Next, participants entered the chamber (40°C, 40% relative humidity) and sat for a 20-minute equilibration period allowing skin temperature to stabilize. During this time, participants filled out an Environmental Symptoms Questionnaire to determine symptoms of heat illness.[51] Each participant had small skin temperature buttons (Thermochrom DS1921G, Measurement Specialties, Beavercreek, OH) taped to his deltoid, chest, calf, and thigh to approximate mean skin temperature throughout the exercise bout. A heart rate strap (RaceTrainer™, Timex Group USA, Middlebury, CT) was fitted to the participant. Resting skin temperature (T_{SK}), rectal temperature (T_{REC}), and heart rate (HR), were recorded. Immediately prior to exercise commencement and every 10 minutes throughout exercise, T_{SK}, T_{REC}, HR, OMNI, and Thirst, Thermal, and Fatigue scales were recorded. The OMNI scale measured perceived exertion using an 11-point scale ranging from 0 (extremely easy) to 10 (extremely hard) in 1.0 increments (Appendix A).[52] Thirst sensation was rated on an 9-point scale ranging from 0 (not thirsty at all) to 9 (very, very thirsty) in 1.0 increments (Appendix B). Similarly, thermal sensation was rated on a 17-point scale ranging from 0 (unbearably cold) to 8 (unbearably hot) in 0.5 increments (Appendix C).[53] Fatigue sensation was rated on a 11-point scale ranging from 0 (no fatigue at all) to 10 (completely fatigued) in 1.0 increments (Appendix D). Participants performed treadmill exercise at approximately 45% VO_2 max at 2% grade, as determined by the baseline

VO_2 max test, for two 60-minute bouts with approximately 13-minute seated rest following each exercise bout. Five minutes into the first HST, VO_2 was checked to ensure participants were exercising at the appropriate speed and adjustments to speed were made at this time if needed. These adjustments were maintained throughout all remaining HST. Additionally, at minute 30 of each exercise bout, VO_2 was calculated for a 5-minute period. Participants drank ad libitum throughout the entire trial. Exercise was terminated if one of the following occurred: 1) T_{REC} reached 40.0° C, 2) HR remained above 90% of age predicted maximum for a 5 minute period, 3) subject volitional exhaustion, or 4) unsteady or unsafe walking gait, 5) 60 minutes of exercise. During the seated rest periods following each exercise bout, participant's grip strength was assessed before and after a 10-minute cooling period. Grip strength measures were obtained via the method previously described above. During the 10-minute cooling period, each participant placed his non-dominant hand in the hand cooling device (CoreControl™, AVAcore Technologies, Ann Arbor, MI) to free his dominant hand for drinking water. When on, the hand cooling device circulated cold water (16.4^{o}C) around the hand while maintaining sub-atmospheric pressure (-40mm Hg) and an airtight seal around the forearm. During the 10-minute period, HR and T_{REC}, were recorded every 2 minutes. Upon completion of the trial, participants exited the environmental chamber and a nude body mass was measured as previously described. Participants were given a clean urine cup for a post exercise urine sample to be collected in privacy, and removed the rectal probe.

Participants performed a total of four heat tolerance tests (HST) in a randomized, crossover fashion. For HST1 and HST3, participants were randomly assigned to either a hand cooling or control trial and performed the opposite trial for HST2 and HST4. During cooling trials, the hand cooling device was on. During the control trial, the participant's hand was in the

hand cooling device, and the device was off. A blood draw was obtained pre and post HST on control days only in order to measure plasma volume change.[54] After sitting still for 10 minutes, a blood draw was performed via an antecubital vein using an aseptic technique by a researcher trained in phlebotomy. Approximately 10mL of blood was drawn, placed on ice, and immediately processed to determine hematocrit and hemoglobin (HB 201+Hemocue Lake Forest, CA) measures. Hematocrit was measured using heparinized microcapillary tubes and centrifuged for 10 minutes (IEC MB Centrifuge, Damon IEC Division, Needham Heights, MA). All samples were run in duplicate. Each HST occurred at approximately the same time of day and was followed by at least one day of rest to ensure the previous day did not influence the following HST. Following HST2, there were 5 days rest to ensure heat exposure during that period did not influence HA.

Heat Acclimation Days

Participants were instructed to drink 500mL water the night before and 250mL the morning of each visit to ensure proper hydration. Upon arrival to the lab wearing a T-shirt and shorts, a urine sample was collected in a clean urine cup to determine urine specific gravity and urine color. Euhydration was defined as urine specific gravity ≤ 1.020. If participants were hypohydrated, they were given approximately 500mL water to consume prior to the start of exercise. Participants privately inserted a flexible rectal thermometer 10cm past the anal sphincter. Nude body mass was obtained in a private location with the participants alone on a scale behind a closed door, with the researcher reading the display outside of the room. Change in body mass from pre and post exercise was used to determine sweat rate during the session. Next, participants entered the chamber (40°C, 40% RH). A heart rate strap was fitted to the subject. Resting T_{REC} and HR were recorded. Prior to exercise commencement and every 15

minutes throughout exercise, HR, OMNI, and Thirst, Thermal, and Fatigue perceptual scales were recorded. T_{REC} was recorded every 5 minutes.

Participants completed between 90 and 240 minutes of treadmill and/or stationary cycling exercise at various intensities. Water was provided *ad libitum*. The goal of each HA day was to achieve a T_{REC} > 38.5°C for a minimum of 60 minutes.[55] Exercise termination criteria were the same as for HST. After completing each exercise bout, participants left the environmental chamber, HR strap was removed, nude body mass was recorded, rectal probe removed, and a urine sample collected to assess hydration.

Data Analysis

A 3-way (acclimation x cooling x bout) repeated measures ANOVA was performed for cooling rate and grip strength variables. Significant F values were further analyzed *post hoc* using Fishers LSD. Paired-samples t-tests were used to analyze differences in physiological measures pre and post HA. Friedman analyses were used for all ordinal perceptual variables and are reported as median(mdn)[interquartile range]. Pearson product-moment correlations were used to analyze associations between cooling rate and the following: hand volume, palm surface area, and body mass. Missing data was replaced with group or within-subject means when applicable; otherwise, data was analyzed pairwise. All data were analyzed using SPSS version 20.0 (IBM Corporation, Champaign, IL, USA) with an *a-priori* alpha level of 0.05. Values are presented as mean±SE.

CHAPTER IV

RESULTS

Participant demographics can be seen in Table 2.

Table 2. Participant Demographics. (mean±SE)

n	Age (y)	Height (cm)	Mass (kg)	VO_2 max (ml/kg/min)	Body Fat (%)	Body Surface Area (m^2)
17	23±1	179.5±1.6	75.30±2.27	54.1±1.3	10.0±1.0	1.93±0.03

Physiological and Perceptual measures demonstrating achievement of HA

Upon HA, T_{REC} for bout 1 (38.20±0.07°C) and bout 2 (38.66±0.15°C) was lower when compared to the respective bout 1 (38.52±0.14°C) and bout 2 (39.21±0.18°C) when NHA ($p \leq 0.05$, Fig. 11). Similarly when HA, HR for bout 1 (135±4bpm) and bout 2 (140±5bpm) was lower than the respective bout 1 (149±6bpm) and bout 2 (158±5bpm) NHA ($p < 0.001$, Fig. 12).

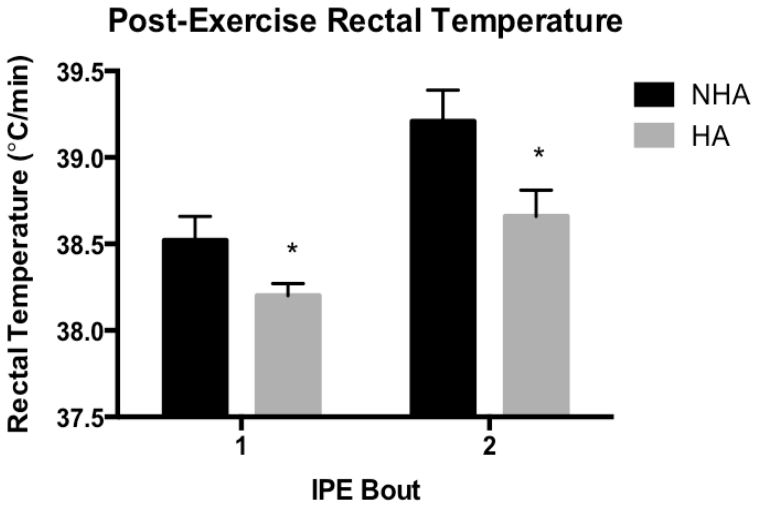


Figure 11. Rectal temperatures following each bout of exercise, before and after heat acclimation. IPE=Immediate post-exercise. NHA=Not heat acclimated. HA=Heat acclimated. Data is presented as mean±SE. * Significant difference compared to NHA, $p \leq 0.0$

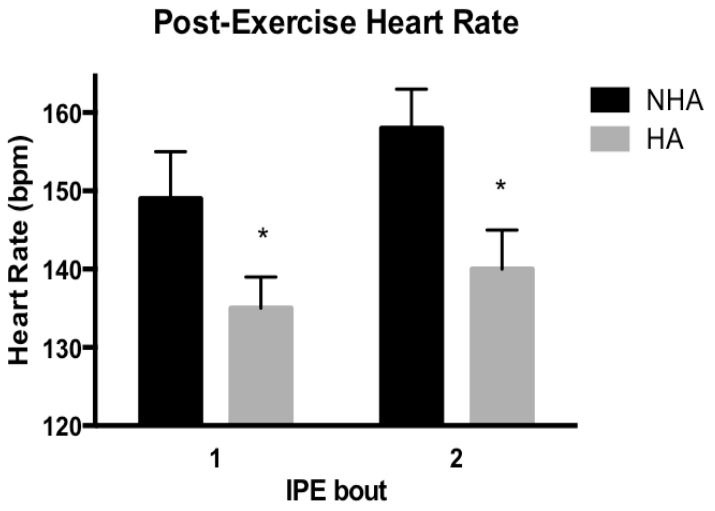


Figure 12. Heart rates following each bout of exercise, before and after heat acclimation. IPE=Immediate post-exercise. NHA=Not heat acclimated. HA=Heat acclimated. Data is presented as mean±SE. * Significant difference compared to NHA, p≤0.05.

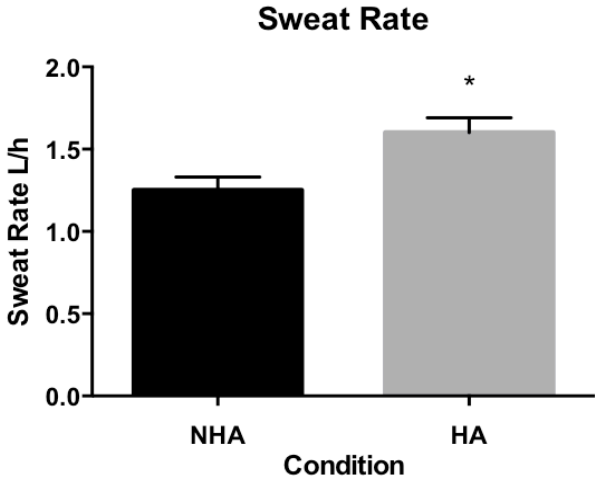


Figure 13. Sweat rates before and after HA. Data is presented as mean±SE. NHA=Not heat acclimated. HA=Heat acclimated. *Significant difference compared to NHA, p≤0.05.

Further confirming acclimation, HA sweat rate (1.60±0.09mL/min) was greater compared to NHA (1.25±0.08mL/min) ($p<0.001$, Fig. 11). IPE OMNI ratings decreased following HA

(mdn=4[5,1]) compared to NHA (mdn=8[4,10]) (p=0.001). Plasma volume change (post-pre exercise) did not differ before (-7.48±1.22%) and after (-7.85±1.19%) HA (p=0.82).

Hand cooling improved cooling rate in NHA conditions only

A significant cooling x acclimation interaction occurred (p=0.02, Figure 14). When NHA, hand cooling had a greater cooling rate (0.020±0.003°C/min) compared to no cooling (0.013±0.003°C/min) (MD [95%CI], p value; 0.007°C [0.001,0.013], p=0.035). Once HA, hand cooling (0.021±0.002°C/min) had a slightly lower, yet not-significant, cooling rate compared to no cooling (0.025±0.002°C/min) (0.004°C [-0.003,0.011], p=0.216). Figure 15 illustrates change in T_{REC} for each 10-minute cooling period, following exercise bouts 1 and 2.

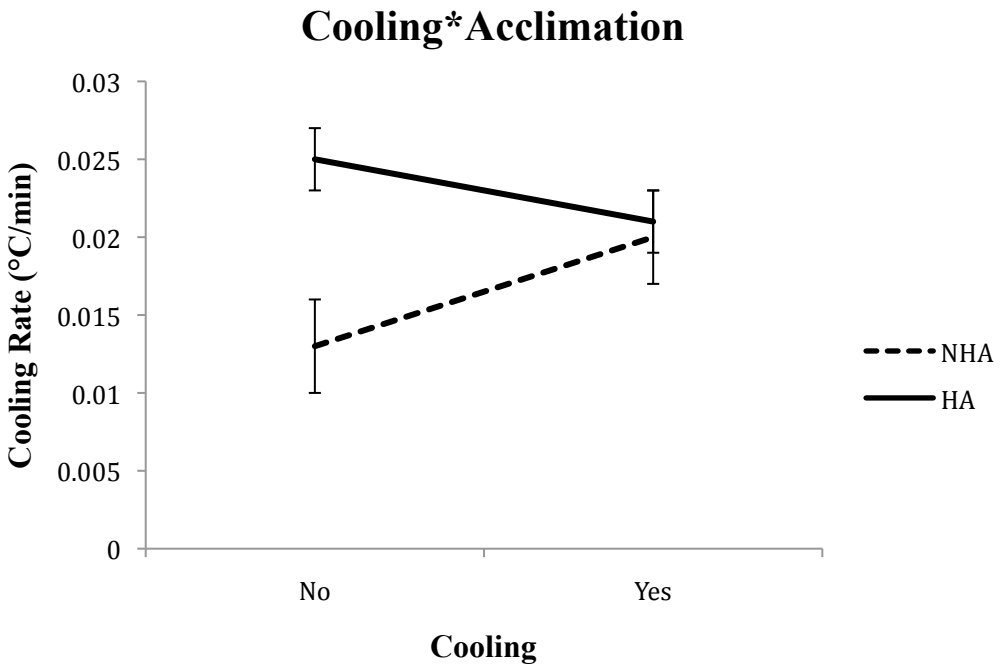


Figure 14. Cooling x Acclimation interaction for cooling rate. NHA=Not heat acclimated. HA=Heat acclimated. Data is presented as mean±SE.

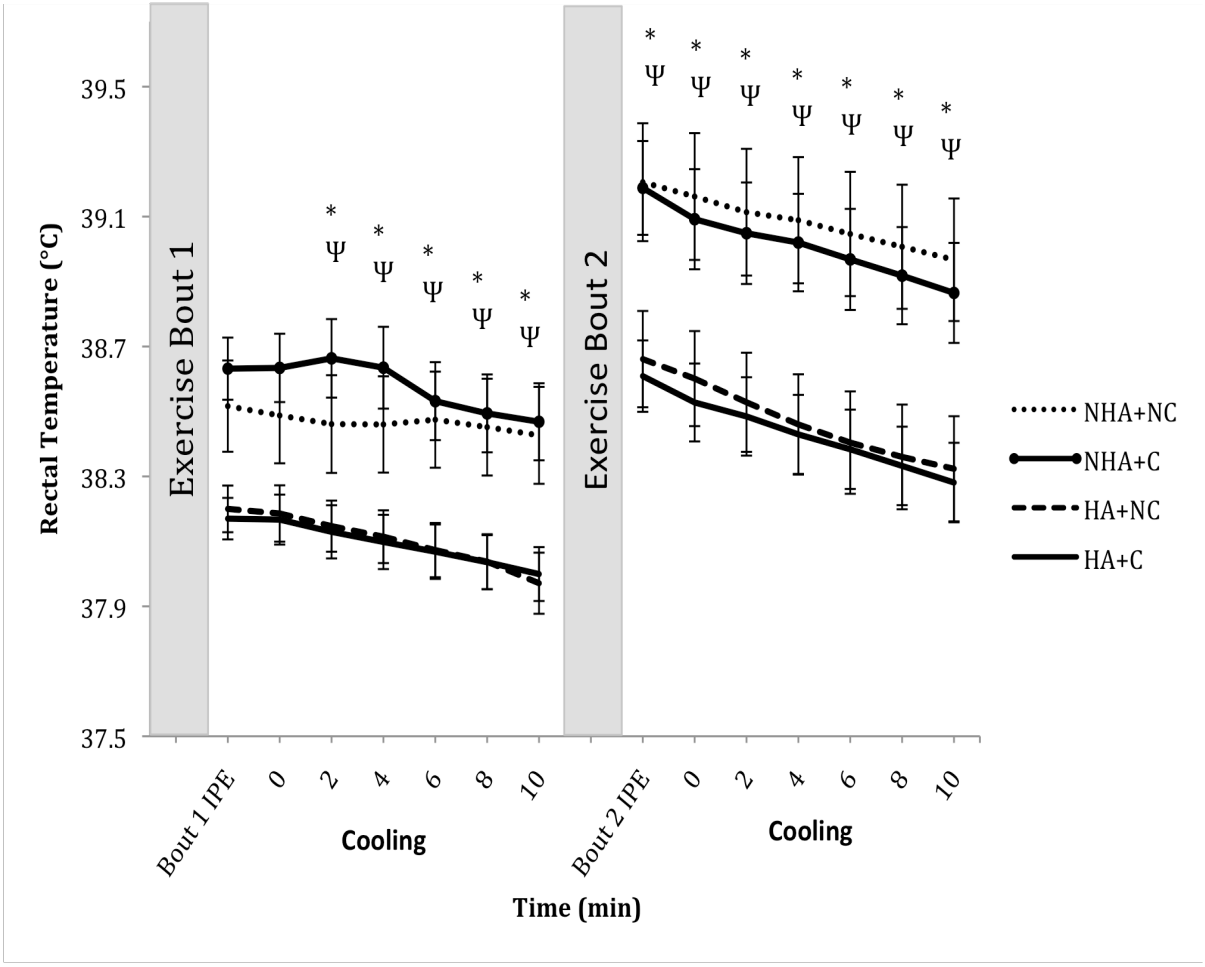


Figure 15. Rectal temperatures during each cooling bout. Exercise bouts were a maximum of 60 minutes. NHA=Not heat acclimated. HA=Heat acclimated. NC=No cooling. C=Cooling. *Significant difference in NHA+NC to HA+NC. $^{\Psi}$Significant difference between NHA+C and HA+C. Data is presented as mean±SE.

HA lowered T_{REC} rate of rise during exercise

When HA, participants had a lower T_{REC} rate of rise (0.019±0.001°C/min) compared to NHA (0.022±0.001°C/min) (0.003°C/min [0.001,0.005], p=0.009). Across all HST, independent of acclimation or cooling status, participants had a lower T_{REC} rate of rise during bout 2

(0.014±0.001°C/min) compared to bout 1 (0.026±0.001°C/min) of exercise (0.012°C/min [0.009,0.014], p<0.001). Cooling had no effect on T_{REC} rate of rise (p=0.121). Table 3 illustrates starting and IPE rectal temperatures as well as rates of rise during each bout of exercise, across all conditions.

Table 3. Rectal temperature rate of rise during exercise.

Condition	Rate (°C/min)	MD[95%CI]	p-value
Acclimation			
HA	0.022±0.001	0.003 [0.001,0.005]	0.009
NHA	0.019±0.001*		
Bout			
Bout 1	0.026±0.001	0.012 [0.009,0.014]	<0.001
Bout 2	0.014±0.001Ψ		
Cooling			
Hand Cooling	0.020±0.001	-0.002 [-0.003,0.000]	0.124
No Cooling	0.021±0.001		

*Significant difference compared to HA. ΨSignificant difference compared to bout 1.

Heat acclimation and hand cooling reduce hand grip strength

Grip strength measures of the dominant hand showed no difference across any conditions (p>0.05). Measures of the non-dominant hand, the hand cooled, indicated a significant acclimation x cooling interaction (p=0.032, Figure 16). When NHA, there was no change (post–pre cooling) in grip strength with (-0.41±0.49kg) or without (-0.68±0.39kg) hand cooling (-0.24kg [-1.74,1.27], p=0.75). Once HA, there was a decrease in grip strength after hand cooling (-2.12±0.74kg), when compared to no cooling (-0.12±0.68kg) (2.00kg [0.31,3.69], p=0.023).

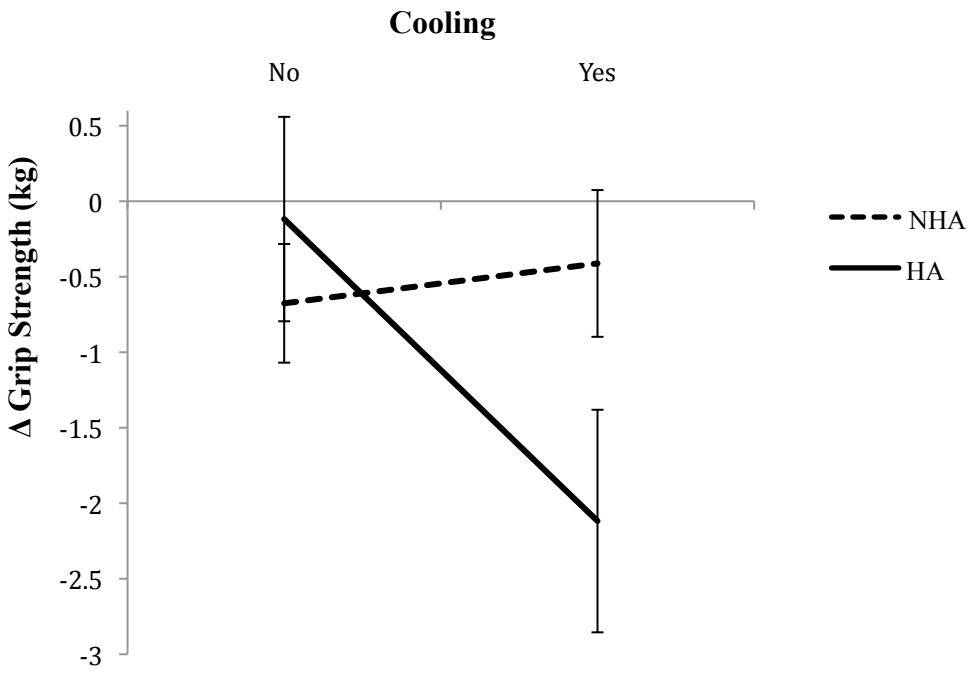
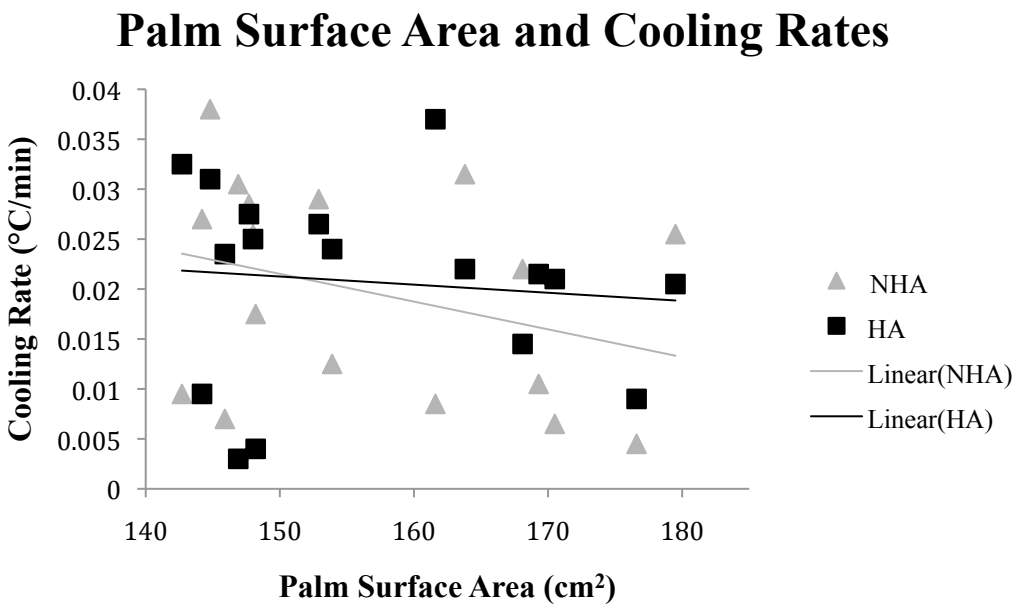


Figure 16. Acclimation x Cooling interaction for grip strength. Δ=post–pre exercise. NHA=Not heat acclimated. HA=Heat acclimated. Data is presented as mean±SE.

Palm surface area and hand volume have no relationship to cooling rate

There were no significant correlations between palm surface area and cooling rate when NHA (r^2=0.103, n=17, p=0.21) or when HA (r^2=0.011, n=17, p=0.70). (Figure 17A) When NHA, a significant correlation between hand volume and cooling rate occurred (r^2=0.264, n=17, p=0.04), and when HA (r^2=0.056, n=17, p=0.36) there was no significant relationship. (Figure 17B)

A.



B.

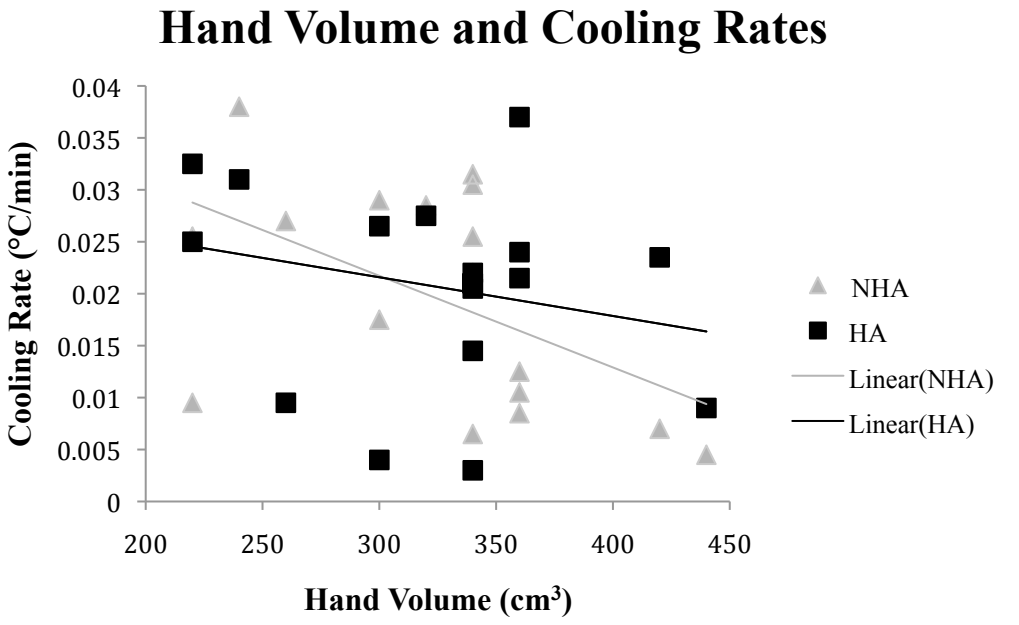


Figure 17. A. Relationship between palm surface area and cooling rate. **B.** Relationship between hand volume and cooling rate.

Perceptual measures

To examine the effects of heat acclimation on perception, NHA+NC and HA+NC measures were compared, as well as NHA+C and HA+C. Results can be found in Table 4. Heat acclimation affected all perceptual measures; however, cooling had no influence on any perceptual measure when NHA or when HA ($p>0.05$).

Table 4. Immediate post exercise perceptual measures across all conditions.

Condition	NHA+NC	NHA+C	HA+NC	HA+C
OMNI	6 [3.5,9]	7 [5,8]	4 [1.5,5]*	4 [2,6]$^{\Psi}$
Thirst	4 [2,5]	4 [3,6]	2 [2,3]*	2 [2,3.5]$^{\Psi}$
Thermal	7 [6,7.5]	7.5 [6.5,7.5]	5.5 [5,6.5]*	6 [5.5,6.5]$^{\Psi}$
Fatigue	7 [3.5,9]	8 [4,8.5]	3 [1.5,6]*	3 [2,5.5]$^{\Psi}$

Data is presented as mdn[IQ range]. NHA=Not heat acclimated. HA=Heat acclimated. NC=No cooling. C=Cooling. *significant compared to NHA+NC ($p\leq0.003$). $^{\Psi}$significant compared to NHA+C ($p\leq0.002$).

CHAPTER V

DISCUSSION

In this study, our aim was to examine the separate and combined effects of heat acclimation and hand cooling efficacy, during and after exercise in the heat. To achieve this, we measured the cooling rate of the CoreControl™ hand cooling device, both before and after a period of heat acclimation. We conclude that when compared to passive rest, hand cooling improved cooling rate when NHA; however, when subjects were HA, it provided no additional benefit.

Previous literature has studied the CoreControl™ hand cooling device in the realm of core body temperature, exercise duration, and work volume during resistance training.[35-39] To our knowledge, this is the first study to quantify the influence of heat acclimation on the efficacy of this device. In order to fully investigate this research question, we had to ensure participants underwent the physiological adaptations of heat acclimation. Upon a period of repeated heat stress exposures, our results show that participants exhibited a lower rectal temperature, heart rate, and perceived exertion, as well as an increased sweat rate, following exercise in the heat. These physiological and perceptual changes are consistent with past literature quantifying heat acclimation and show that our protocol successfully induced appropriate and expected adaptations.[19,56]

Of the limited studies utilizing this hand cooling device, less than optimal cooling rates have been reported (e.g. 0.015-0.040°C/min) .[35-39] Our data showed cooling rates of 0.020-0.021°C/min, thus supporting these past findings. Compared to other cooling modalities such as fans plus shade or cold water immersion with cooling rates ranging 0.11-0.25°C/min,[40] the cooling efficacy of the CoreControl™ device remains far inferior. We attribute its minimal

cooling ability to a few reasons, most notably, limited venous return. Of the number of factors impacting venous return, the two most pertinent to the context of our study include muscular contraction and elevation. When individuals are cooling with this device, blood is being drawn to the hand via negative pressure; however, there is neither muscular contraction nor elevation within the hand to allow for efficient venous return. This was indicated by the edema our participants experienced, and as past literature suggests, peripheral edema causes an increase in interstitial fluid, thus compressing peripheral veins, elevating mean systemic pressure, and ultimately reducing venous return.[41] Additional explanations for low cooling rates of this device included a small surface area being cooled, as well as its lack for targeting major arteries. The cooling potential of this device was calculated empirically by comparing temperature changes in containers of water, one with the head cooling device and one without. Using the specific heat of water (4. J), we have calculated the cooling potential to be 11W. Relatively, with a cooling surface area of $0.02m^2$, this device cools at $55W/m^2$. Compared to other cooling devices such as ice-pack head or vest devices, this cooling potential is far inferior.[57]

Interestingly, when participants were NHA, hand cooling provided greater cooling rates compared to passive rest. Rectal temperature decreased at a rate of 0.020°C/min, in comparison to 0.013°C/min when no cooling was implemented. While this mean difference of 0.007°C/min provided statistical significance, in a clinical setting, the cooling rate of this device is far from ideal. At a rate of 0.020°C/min, it would take 50 minutes to decrease rectal temperature 1°C. When individuals are not fully HA (e.g. first few days of a HA protocol), utilizing the CoreControl™ device would provide a greater decrease in rectal temperature, beyond that of passive rest; however, given this device's characteristics (e.g. size and cost), other cooling modalities may be more practical. Future studies should examine this device in relation to other

cooling options (e.g. ice towels, fan, etc) to determine if its cooling efficiency is worth the cost. Given that exertional heat illnesses, especially exertional heat stroke, most commonly occur when individuals are not heat acclimated (e.g. first few days of a novel heat stress), it is crucial that we fully optimize cooling potential during this time in hopes of preventing these life-threatening conditions. Our data supports the notion that cooling when NHA is beneficial; however, other cooling modalities that may provide greater cooling rates should be studied in this context.

Once participants achieved HA, the benefits of this device were diminished. Hand cooling rates of 0.021°C/min were observed in HA individuals, and were lower, yet not significant, compared to passive rest (0.025°C/min). Given this minimal and insignificant difference, we conclude no synergistic benefits were obtained with hand cooling once HA. Most importantly, passive cooling when HA (0.025°C/min) was greater than both passive rates (0.013°C/min) and cooling rates (0.020°C/min) when NHA. These results provide supportive evidence for the benefits of HA; showing that, within the context of our study, HA provided the most optimal thermoregulation and heat dissipation potential.

Further investigation for benefits of cooling led us to examine rectal temperature rate of rise during each bout of exercise. While cooling provided no reduction in rate of rise, a reduced overall rate of rise was observed in HA individuals (0.019°C/min) compared to when NHA (0.022°C/min). This difference, while minimal, also significantly contributes to the evidence on the well-established benefits of HA. A reduced rate of rise contributes to an overall decrease in core body temperature during and upon completion of exercise. This is important for the attenuation of body temperatures and prevention from reaching hyperthermic levels, ultimately aiding in reduced chances for heat illnesses.

The secondary aims of our study were to examine the influence of hand cooling on grip strength and to explore the relationship between cooling rate on both palm surface area and hand volume. To achieve this, we measured grip strength before and after each hand cooling period, as well as obtained hand size and palm surface area measurements at baseline. Our results conclude that grip strength decreased following cooling, but this was only observed after individuals were HA. Additionally, there were no significant correlations between cooling rate and both palm surface area and hand volume indicating no relationship between these variables.

Previous literature examining the effects of hand cooling on resistance exercise show the CoreControl™ increased bench press strength when utilized between sets.[42,43] This led us to hypothesize that hand cooling would also improve grip strength. Conversely, we found hand cooling to have decreased hand grip strength in the cooled, non-dominant hand under all conditions, however, most significantly when individuals were HA. We believe the decrease in grip strength can be attributed to the edema participant's experienced following use of this device. Additionally, cooling the hand may have led to an internal muscular temperature below that for which is optimal for maximal strength within the hand. Since the hand that was not cooled (dominant hand) did not experience significant changes in grip strength, we can attribute the decrease in grip strength to the cooling device. The rationale as to why hand cooling decreased grip strength to a larger extent in only HA conditions is far more puzzling. Physiologically, when HA, increased blood flow to the periphery along with decreased perceived exertion, would lead us to assume the opposite that a greater decrease in strength would be observed when NHA rather than HA. Given this is the first study to examine grip strength following hand cooling, future studies should be conducted to see if similar results are achieved.

Novel to this study is the examination between cooling rates and both palm surface area and hand volume. We hypothesized that larger hands (e.g. greater surface area and greater volume) would result in higher cooling rates, given more of the body was being cooled. No statistical correlation was found between these variables indicating no relationship between hand size and cooling rate. It is likely that no relationship was found due to the large variability and small sample size of this study.

Overall, hand cooling using the CoreControl^{TM} device provided a greater cooling rate compared to passive rest when individuals were NHA, and upon HA, this device showed no benefit. Therefore, this device is useful only when individuals are NHA, however, cooling rates still remained minimal. In the context of this study, cooling rates were greatest when passively cooling once HA. Given the many benefits of HA, including its ability to reduce incidences of heat illnesses,[13,14] it is strongly recommended individuals first and foremost achieve HA. Additionally, hand cooling decreased hand grip strength once individuals were HA, further confirming its very limited advantages. Finally, there were no relationships between hand volume and palm surface area on cooling rate. In conclusion, our results show that the CoreControl^{TM} hand cooling device has limited benefits and should not be used as a modality for decreasing body temperature quickly.

CHAPTER VI

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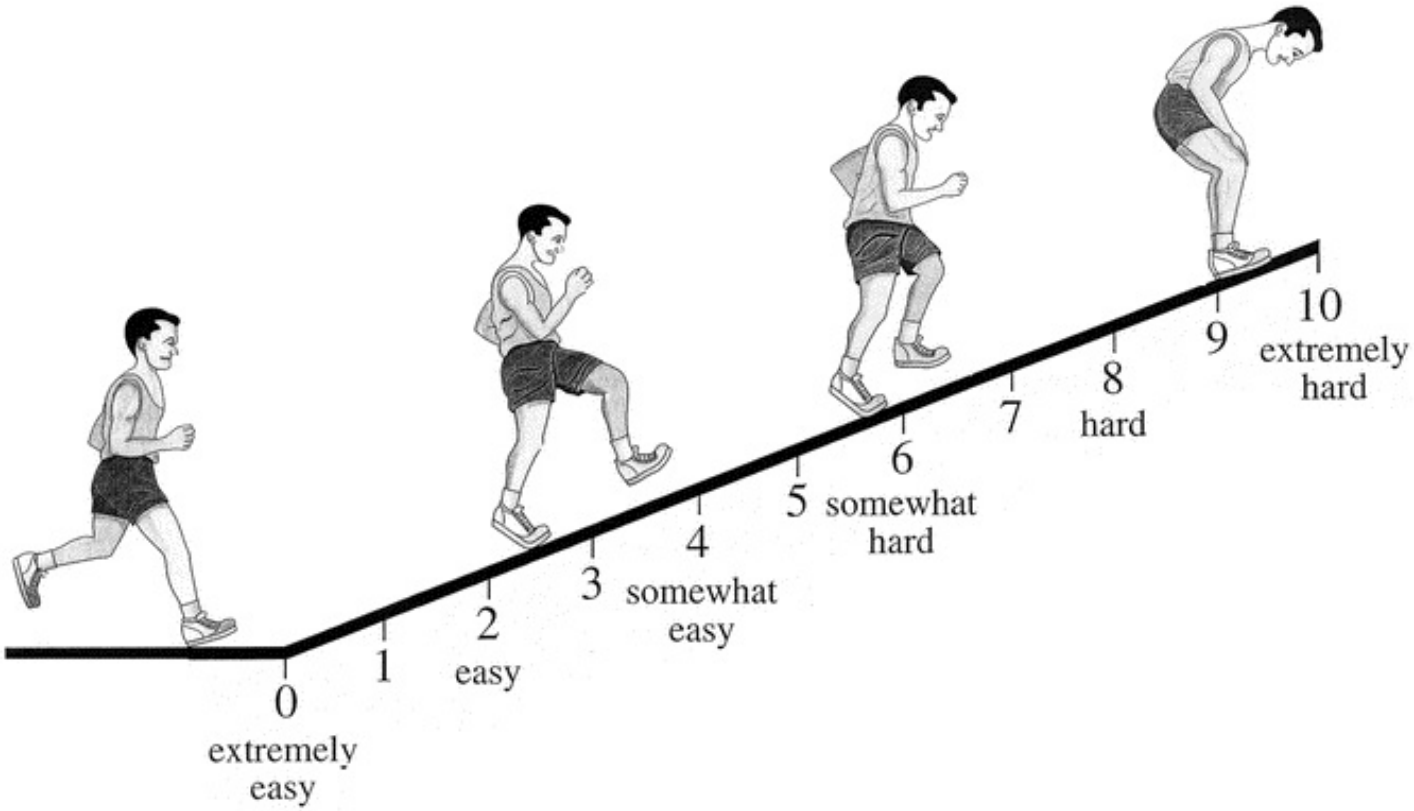
APPENDIX A

OMNI Scale



APPENDIX B
Thirst Scale

1 **Not Thirsty At ALL**

2

3 **A Little Thirsty**

4

5 **Moderately Thirsty**

6

7 **Very Thirsty**

8

9 **Very, Very Thirsty**

APPENDIX C
Thermal Scale

0 Unbearably Cold

1 Very Cold

2 Cold

3 Cool

4 Comfortable

5 Warm

6 Hot

7 Very Hot

8 Unbearably Hot

APPENDIX D
Fatigue Scale

INDICATE YOUR LEVEL OF OVERALL FATIGUE <u>RIGHT NOW</u>

0 No Fatigue At All

1 Very Small Amount of Fatigue

2 Small Amount of Fatigue

3 Moderately Fatigued

4 Somewhat Fatigued

5 Fatigued

6

7 Very Fatigued

8

9 Extremely Fatigued

10 Completely Fatigued